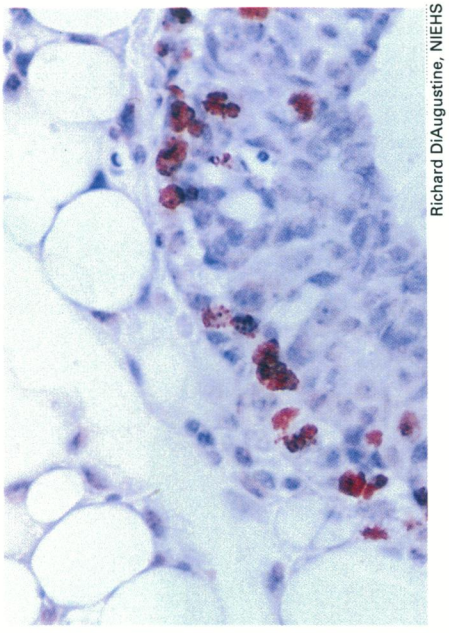
In the not-distant future, there will be a new "sacred agenda" in international affairs: policies that enable rescue of the global environment. This task will one day join, and even supplant, preventing the world's incineration through nuclear war as the principal test of statecraft.
Albert Gore, speech, Global Change Conference, Smithsonian Institution and the National Academy of Sciences, 3 May 1989

Forum

Comprehensive Strategies Needed to Study Breast Cancer

Breast cancer strikes one out of every nine women in the United States, and there is evidence that these rates have been steadily increasing over the last 20 years. Scientists are now taking a closer look at studies which indicate that breast cancer is produced by a complex interaction of such factors as hormonal status, genetic susceptibility, environmental exposures, and dietary components.

Results of several studies conducted by Japanese and American researchers show that breast cancer rates in Japan are low (1 in 50), but rates in Japanese-American women approach those present in the U.S. population, indicating that other factors in addition to genetics play a part in whether a woman will develop breast cancer. A study reported in the *American Journal of Epidemiology* (volume 134, 1991) focusing on the effect of smoking on breast cancer rates indicated that, although there does not appear to be an increased risk of breast cancer in women who begin smoking as adults, breast cancer risks are significantly elevated in women who begin smoking as teenagers. Similarly, a study of atomic bomb survivors from Japan revealed that adult women exposed to radiation had breast cancer rates similar to controls, but teenage girls exposed to those same levels had markedly greater breast cancer rates later in life. These findings suggest that susceptibility to cancer-promoting environmental exposures may also be age dependent.



Richard DiAugustine, NIEHS

Development of breast cancer. Rapid cell division (indicated by red staining) in the developing mouse mammary gland enhances the potential for chemically induced mutations.

Animal studies bear out these findings as well. Thirty-two years ago, a study by Nobel Prize winner Charles Huggins showed that administration of polycyclic aromatic hydrocarbons (PAHs) to 50-day-old female rats produced nearly a 100% incidence of mammary cancer, as opposed to a much lower incidence in younger or older rats exposed to the same doses of these chemicals. PAHs are found in urban air pollutants and cigarette contaminants. Scientists believe that exposure to carcinogens during the period of rapid proliferation of mammary cells during puberty may permit fixation of DNA damage in the genome. Subsequent exposure to ovarian hormones, such as estradiol and progesterone, which stimulate proliferation of normal breast cells, may also expand this damage, resulting in the formation of breast tumors. Evidence shows that the incidence of breast cancer is markedly reduced in women who have had their ovaries removed before 35 years of age.

In addition to the interplay among environmental exposures, genetic susceptibility, and hormonal factors, many scientists now add diet as a possible indicator of risk for breast cancer. Although this contention is widely debated, some studies have indicated a possible link between levels of dietary fat and caloric intake and the incidence of breast cancer.

The catastrophic magnitude of breast cancer is compelling researchers to develop research strategies aimed at understanding the integrated roles of all of these factors in causing and promoting breast cancer so that effective prevention and intervention approaches can be developed.

New EPA Administrator Enters Debate over Pesticide Residues in Processed Foods

Within two weeks of the confirmation of her appointment as administrator of the Environmental Protection Agency, Carol M. Browner was faced with difficult policy decisions regarding pesticide residues in processed foods.

The EPA administers the section of the Federal Food, Drug and Cosmetic Act containing the "Delaney Clause" that prohibits any food substance that contains any cancer-causing pesticide from being processed for human consumption. The Delaney Clause was added to the Act in 1958 by the late New York Congressman James Delaney after congressional investigation of chemicals in foods and cosmetics. It was intended to protect consumers from additional cancer risks presumed to result from consumption of contaminated foods. The clause has remained in the Act ever since.

In the intervening years, analytic chemistry methods and techniques have improved dramatically, and pesticide residues can now be detected at levels below one part per trillion. At the same time, toxicologic studies have been conducted that demonstrate some level of carcinogenic activity for many agricultural chemicals. Under the Delaney Clause, no food products containing any amount of a compound that has tested positive in any laboratory study may be present in processed food. Many agricultural interests and pesticide manufacturers argue that the Delaney Clause is too restrictive and should be relaxed to permit very low levels of certain pesticides in processed foods, particularly when the toxicologic evidence of carcinogenicity from animal studies is equivocal or weak. The suggestion that "*de minimis*" levels be established for such pesticides gained support in a National Academy of Sciences report in 1989.

There is precedent for the *de minimis* approach. The Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) allows EPA to establish permissible trace residues of chemicals that are carcinogenic in toxicologic studies in nonprocessed,